THE SYMPTOMS OF AIR POLLUTION INJURIES TO BROAD-LEAVED FOREST TREES

by

Leon S. Dochinger,
Principal Plant Pathologist
United States Department of Agriculture
Forest Service
Northeastern Forest Experiment Station
Forest Insect and Disease Laboratory
Delaware, Ohio 43015, U. S. A.

ABSTRACT

The impact of air pollution on forest tree plantings is of intense concern throughout the world. Many air-pollution problems can be resolved only by international programs of cooperative research. Because of the apparent sensitivity of and more conspicuous injuries to conifer trees by air pollutants, little has been accomplished in delineating the symptomatology of hardwood trees. The most important tree pollutants are sulfur dioxide, fluoride, and ozone. Attempts to relate these aerial contaminants to effects on hardwood trees are hampered by deficiences in instrumentation and methods of atmospheric analyses, by inadequate knowledge about how various factors alter comparatively simple gaseous forms into phytotoxicants of indefinable composition, by the inability to distinguish air-pollution symptomatology from that caused by other causal agents, and by insufficient information about changes and interactions with which air pollutants are influenced by the total environment. Investigations are needed to characterize acute and chronic symptoms caused by single and multiple pollutants and to relate pollution injury to the modifying conditions of environmental parameters upon forest hardwood trees. If our outlook remains unchanged toward curbing the harmful aspects of air pollution as our population spirals upwards, as demands for energy increase, and as more wastes are released into the atmosphere, we may experience injury and damage to forest trees of a nature and magnitude not yet imagined.

INTRODUCTION

Air pollution is neither a modern day phenomenon nor a local, indigenous problem. Contamination of international airsheds has slowly intensified along with the evolution of the human race and the use of fire to provide for energy needs. But in current times, man has intensified the insidious fouling of the atmosphere through his exploding increase in global population, his insatiable requirements for generation of power, and his long term disregard for adequate methods to control aerial wastes harmful to the earth's vegetation. If the cleansing nature of our atmosphere is not improved, the survival of civilization may be in jeopardy. A program must be initiated for the rational use and management of the atmosphere by all nations concerned in safeguarding and rehabilitating our contaminated atmosphere.

Forest tree plantings are injured by air pollutants primarily through their foliage. Conifer species are more prone to injury, because their needles are retained for several years. In contrast, hardwood trees bear their foliage only a portion of the year, shed their leaves, and reproduce a new crop of leaves annually.

Because of the more conspicuous symptoms associated with air pollution effects on conifer trees, only a limited research effort has been given to delineating the symptomatology of pollution injury to broad-leaved trees. I will discuss the relation of the air pollution syndrome of deciduous trees in forest plantings to sulfur dioxide, fluorides, and ozone, and some of the research needs for further elucidation of injury to forest hardwood species by aerial phytotoxicants.

An air pollutant may be considered as an aerial substance that produces an unfavorable response on materials, vegetation, or man. These atmospheric contaminants may include materials the consistency of soil particles, radioactive dust, salt spray, herbicide and pesticide aerosols, liquid droplets of acidic matter, gases, or all of these natural and artificial materials in various combinations. Therefore, an aerial phytotoxicant may be any airborne constituent supplied in a sufficient concentration for a specified period of time to alter the physical or chemical properties of other environmental components sufficiently to cause a harmful effect on plant life.

Air pollutants are classified into a variety of categories. Perhaps the most convenient taxonomic system is to separate pollutants into two general groups: (1) primary emissions which originate from an identifiable source in a form toxic to vegetation, such as sulfur dioxide and fluoride compounds; and (2) secondary pollutants produced in the atmosphere by combinations of primary toxicants, reactions with indigenous aerial materials, or through photolytic activation. The latter includes harmful combinations between metallic compounds and water vapor and the photochemical pollutants of ozone and the homologous series of peroxyacetyl nitrate and its relatives.

Innumerable air pollutants are responsible for toxic effects on vegetation. Although their relative importance as phytotoxicants vary from location to location, it is apparent from international estimates of the economic damage in forest and natural tree plantings that the primary offenders are sulfur dioxide, fluorides, and ozone.

SULFUR DIOXIDE

Sulfur dioxide effluents from industrial complexes are considered a major cause of pollution injury to forest plantings throughout the world. More investigations were conducted in the last 100 years on the effects of this gaseous pollutant upon vegetation than on any other pollutant.

In early German publications, Schroeder and Reuss (1883) and Haselhoff and Lindau (1903) reported the manifestations of acute and chronic damage on numerous conifer and deciduous trees. More detailed reviews of the literature of vegetation injury from the oxides of sulfur may be found in the publications by Thomas (1951 and 1956), Thomas and Hendricks(1956), Thomas et al. (1952), Brandt and Heck (1968), Daines (1968), Katz and McCallum (1952), Scheffer and Hedgcock (1955), Wentzel (1956), Scurfield (1960), Dassler (1963), and Knabe (1966).

In North America, gross damage to thousands of acres of forest trees were induced by high-level fumigations. Detailed studies of smelter fume damage were reported from Redding, California (Haywood 1905); Anaconda, Montana (Hedgcock 1912); Ducktown and Copper Hill, Tennessee (Hedgcock 1914; Scheffer and Hedgcock 1955); Trail, British Columbia (Katz et al. 1939); and Wawa, Ontario (Gordon and Gorham 1963).

More recent reports of severe damage to forests near industrial sites in European and Asian countries come from Western Germany (Wentzel 1961), Great Britain (Scurfield 1960), Czechoslovakia (Nozicka 1963, Dimitrovsky 1965), France (Bossavy 1965), Hungary (Berend and Csuti 1959), Russia (Antipov 1957), and Japan (Okanoue 1958).

Sulfur dioxide originates from the combustion of nearly all fuels, especially coal and oil, and from the heating of sulfide ores during smelting procedures. Other main sources are from the production and utilization of sulfur, sulfuric acid, petroleum, and natural gas. Combustion of sulfur-containing compounds release vast amounts of sulfur dioxide and some sulfur trioxide into the atmosphere, and industries emit some hydrogen sulfides and mercaptans in their processing operations.

In the atmosphere, sulfur dioxide is measured by colorimetric, conductometric, and coulometric sampling techniques. Both continuous and intermittent sampling are generally used for sulfur dioxide. Recently improved West-Gaeke pararosaniline methods (Scaringelli et al. 1967) were developed for the determination of sulfur dioxide in ambient air. Hochheiser (1964), Altshuller (1965), and Pack and Adams (1966) have published more extensive information on measuring and monitoring atmospheric sulfur dioxide.

Symptoms

Sulfur dioxide injury on forest broad-leaved trees can either be acute or chronic. Acute markings supposedly occur after the rapid intake of toxic concentrations of sulfur dioxide. Shortly after exposure, tissues become dull and hydrotic and then they collapse, usually assuming a gray-green color. These affected areas later dry and bleach to a light-tan or ivory color in many species, but occasionally the lesions may be brown or reddish brown. The final pattern of acute injury is either marginal or intercostal with minimum effect across the veins of broad-leaved trees unless injury is quite severe.

Linzon (1969) observed that acute injury on deciduous trees could occur as bifacial lesions between veins. Developing leaves rarely exhibited necrosis and oldest leaves were moderately susceptible; but newly expanded leaves were most sensitive, especially toward the petioles.

Katz et al. (1939) described various markings and discolorations on the foliage of broad-leaved trees resulting from SO_2 fumigation in the vicinity of Trail, British Columbia. He reported the markings were usually intercostal on birch (Betula species), but marginal burn was also common. The intercostal markings consisted of many small brown spots that often coalesced to form elongated regions between the primary veins. On aspen (Populus species), all tissues were destroyed except for a section of midrib. On lightly injured leaves, markings were confined to side veins. Shortly after fumigation, acute markings were an opaque, reddish brown, and these necrotic spots blackened later in the year. Maple leaves (Acer species) occasionally displayed marginal injury extending deep between the veins of a leaf. Frequently, a dull, hydrotic margin was present between healthy and dead tissues. Often, light-brown intercostal patches were produced. Willow (Salix species) showed diffuse markings, primarily on the upper epidermis. The injury was mostly confined to the midrib in the main veins, rarely appeared on the edge of the leaf, and was a dark, reddish-brown color, which faded out into the green pigment of the foliage. The markings on alder (Alnus species) were akin to birch, being mostly intercostal, often marginal, and penetrating between the side veins. Early injury was light brown; but, in time, the areas became dark red.

The effects of exposure to continuous sublethal concentrations of sulfur dioxide over a considerable period of time are characterized by an incipient yellowing or chlorotic symptom of chronic injury on leaves of forest broad-leaved trees. The gradual decline in green color suggests that chorophyll is destroyed and not resynthesized. Leaves may remain turgid and appear to function at a rate of efficiency proportional to the amount of chorophyll destroyed. Chronic symptoms can be localized into a few flecks of injury varying in color from yellow, ivory, bronze, or black. Some hardwood species exhibit a diffuse mottling on their leaves. Both chronic and acute symptoms may be present on either one or both surfaces of the leaves.

Leaf markings are the typical symptoms on forest broad-leaved species injured by sulfur dioxide. But reduction in growth of hardwood trees may also occur as exhibited on conifer species grown near sources of sulfur dioxide (Scheffer and Hedgcock 1955). The suppression in growth was attributed to premature defoliation and destruciton of needles during prolonged exposures to high levels of sulfur dioxide. Gordon and Gorham (1963) have observed similar retarded growth on broad-leaved forest species in Canada.

Whitby (1939) and Thomas et al. (1950) found large amounts of sulfate in analyses of leaves with chronic symptoms. Acutely injured leaves had only slightly higher sulfate content. High quantities of sulfate did occur in leaf tissues without injury. Yet, if excessive amounts are absorbed quickly, acute injury resulted. Dochinger (1968) clarified some of the inconsistencies between sulfate content and degree of injury in leaves of white pine trees (Pinus strobus L.). He observed that scions from pollution-susceptible trees exhibited the same degree of symptom intensity annually as their parents when grafted to tolerant white pines. And scions from tolerant trees continued vigorous when grafted to sensitive white pines. His findings indicated that tree-to-tree variations in responses to injurious pollutants were genetically controlled by inherited factors.

Mode of Action

Although many explanations were proposed for the injurious action of sulfur dioxide to vegetation, none has totally explained the mechanism of action by this gas. Hasclhoff and Lindau (1903) reported that sulfur dioxide was tied to aldehydes and subsequent degradation by-products of sulfuric or sulfurous acid injured plant tissues. Novak (1929) believed an inactivation of iron in the chloroplasts prevented the assimilation of organic compounds. Dorries (1932) proposed that an interaction of acidic compounds from sulfur dioxide could split magnesium from chlorophyll and form pheophytin. Thomas (1951) attributed acute symptoms to excessive sulfite or sulfurous acid accumulation in plant tissues. Nikolaevskŷ (1968) studied the activity of peroxidase, polyphenoloxidase, and catalase in woody trees. He concluded that inactivation of catalase by sulfur dioxide was accompanied by increases of the other two enzymes, which in turn reduced susceptibility of trees to pollution injury. A recent report suggested that the formation of hydrogen sulfide during and after sulfur dioxide fumigation may be destructive to leaves (Anonymous 1968).

Susceptibility

Numerous investigators, including Thomas et al. (1950), Pelz (1956), Brandt and Heck (1968), Daines (1968), and Dochinger and Seliskar (1970), have presented their findings that different species, varieties within a species, and even clones within a plant variety vary in their susceptibility to sulfur dioxide. Reviews by Setterstrom and Zimmerman (1939), Katz (1949), Zahn (1963), Heck (1968), Wentzel (1968), and Barrett and Benedict (1970) further explain the responses of plants to sulfur dioxide as they are influenced by maturity of tissues, soil moisture, interactions with other aerial contaminants, levels and fluctuations in pollution concentration, relative humidity, stomatal movements, and related environmental factors.

Hedgcock (1914) surveyed the condition of trees in the regions around Ducktown and Copper Hill, Tennessee. Deciduous trees appeared to be more tolerant of sulfur dioxide fumigation than conifers. The hardwoods along the Ocoee River to the west that were more resistant are as follows (the most tolerant being named first): white oak (Quercus alba L.), red maple (Acer rubrum L.), flowering dogwood (Cornus florida L.), blackgum (Nyssa sylvatica L.), eastern cottonwood (Populus deltoides Bartr.), sourwood (Oxydendron arboreum L.), yellow-poplar (Liriodendron tulipifera L.), black locust (Robinia pseudoacacia L.), and American sycamore (Platanus occidentalis L.).

On the uplands, most injury was noticed on the slopes of hills facing smelters. Of the oak trees, the order of resistance was as follows: white oak, chestnut oak (Q. prinus L.), scarlet oak (Q. coccinea Muenchh.), pin oak (Q. palustris Muenchh.), southern red oak (Q. falcata Michx.), post oak (Q. stellata Wangenh.), blackjack oak (Q. marilandica Muenchh.), black oak (Q. velutina Lam.), and southern red oak (Q. falcata Michx. var. falcata).

Scheffer and Hedgcock's (1955) order of tree species susceptibility showed little agreement with that by Katz et al. (1939). They related the differences to the criterion of susceptibility, location, time of year, and injury zone. In the upper Columbia River Valley, Washington, only the forest broad-leaved species were alive near the smelter. Their approximate sequence of susceptibility, beginning with the most sensitive, was thinleaf alder (Alnus tenufolia Nutt.), western paper birch (Betula papyrifera var. commutata (Reg.) Fern), Sitka mountain-ash (Sorbus sitchensis Roem.), water birch (B. occidentalis Hook.), Douglas maple (Acer glabrum var. douglasii (Hook.) Dipp.), bitter cherry (Prunus emarginata Dougl.), common chokecherry P. virginiana L.), blueberry elder (Sambrucus glauca Nutt.), willow (Salix species), Columbia hawthorn (Crataegus columbiana Howell.), black cottonwood (Populus trichocarpa Torr. and Gray.), black hawthorn (C. douglasii Lindl.), and quaking aspen (P. tremuloides Michx.).

In Germany, Pelz et al. (1963) appraised the areas near the Freiberg lead works where the pollutants were mostly sulfur dioxide and arsenic dusts. Within a radius of about 1000 m, samplings of increment growth suggested that birches, alders, and hybrid poplars were tolerant, and oak trees more clearly reflected the periods of high and low releases of noxious substances into the surrounding forest plantings. Prokopiev (1965) inspected 10- to 35-year-old plantings of red oak and English oak (Q. robur L.) for damage by sulfur gases in forests near the G. Damjanov' copper combine in Bulgaria. He determined red oak was more tolerant to sulfur dioxide. Spierings (1967) presented his data on experimental sulfur dioxide fumigation that showed European mountain-ash (Sorbus aucuparia L.) to be very susceptible and European beech (Fagus sylvatica L.) to be fairly resistant.

In Turkey, Acatay (1968) studied the effects of sulfur dioxide damage from the copper smelting works in Murgul woodlands. Hardwoods species, arranged in order of increasing sensitivity, were Durmast oak (Q. petraea Lieblein), English elm (Ulmi procera Salisb.), Black locust, oriental beech (Fagus orientalis Lipsky.), date plum (Diospyros lotus L.), Linden (Tilia species), trembling aspen, European birch (Betula pendula Roth.), alder (Alnus species), hornbeam (Carpinus species), ash (Fraxinus species), European mountain ash, common filbert (Corylus avellana L.), Spanish chestnut (Castanea sativa Mill.), European hop hornbeam (Ostrya carpinifolia Scop.), and Persian walnut (Juglans regia L.).

At Wawa, Canada, Gordon and Gorham (1963) surveyed damage by sulfur dioxide pollution from an iron sintering plant. Hardwood trees most tolerant to pollution were pin cherry (Prunus pensylvanica L.), red maple (Acer rubrum L.), sugar maple (A. saccharum Marsh.), and Mountain maple (A. spicatum Lam.). In quadrat studies along a northeast transect, no trembling aspen seedlings grew within 15 miles of the plant.

Linzon (1965) inspected damaged broad-leaved trees in the vicinity of petroleum refineries located at Clarkson, Ontario, and East St. Paul, Manitoba. Hardwoods with marginally and intercostally damaged foliage included species of birch, poplar, maple, oak, and beech at the former site. At the St. Paul refinery, most severely injured trees were bur oak (Q. macrocarpa Michx.), boxelder (Alnus negundo L.), and trembling aspen. American elm and chokecherry displayed less injury, while balsam poplar (Populus balsamifera L.) and green ash (F. pennsylvanica Marsh.) were only partly affected.

In the Sudbury Basin, Canada, Dreisinger and McGovern (1970) monitored atmospheric sulfur dioxide from nickel smeltering companies and studied damage to forests. Among the forest species, trembling aspen was the most sensitive tree. Foliage of this species and paper birch was extremely sensitive to sulfur dioxide, but was more resistant to repeated fumigations than some of the conifers, especially eastern white pine. The order of ascending tolerance to pollution by the remaining broad-leaved species was bigtooth aspen (Populus grandidentata Michx.), willow, alder, balsam poplar witch hazel (Hamamelis species), red oak, and sugar maple.

FLUORIDES

During the late 19th century, German workers described fluoride damage to vegetation near industrial sites of copper smelters (Schroeder and Reuss 1883), superphosphate production centers (Mahyrhofer 1893; Rhode 1895; and Wislicenus 1898), and glass processing and fertilizer factories (Ost 1907). Further reviews of fluorine damage were made in the European literature by Haselhoff et al. (1932) and Romell (1941).

For the last 30 years, the injury to agriculture by fluorine compounds has intensified because of the expansion of industries involved with aluminum manufacturing, the mining of phosphate deposits, and the production of fertilizers. Recent cases of fluoride injury are those described by Gisiger (1955), Scurfield (1960), Holte (1961), Garber (1963), and Bossavy (1965a). Fluoride pollution is important in North America and has occurred in Tennessee (MacIntire 1952), New Jersey (Daines et al. 1952), and California (Middleton et al. 1965).

Fluorides in minute concentrations are usually present in water, soil, and vegetation; however, it is extremely unlikely that the relatively low natural levels of these compounds are detrimental to plants. Injuries are from the discharges of fluorides into the atmosphere mainly from the gaseous and particulate waste compounds of the following industrial operations or community sources: the smelting of nonferrous ores, steel, and aluminum; combustion of coal; manufacture of brick, ceramics, glass, and phosphate fertilizers; synthesis and utilization of hydrofluoric acid; cement plants; and the use of fluorides as oxidizing agents in rocket fuels.

The primary gaseous forms of fluorides evolved from these processes are hydrogen fluorides and silicon tetrafluorides. The kinds of particulate fluorides emitted by industries are not completely identifiable. It is known that aluminum fluoride and cryolite are formed from aluminum smelting; and from phosphate manufacturing, calcium fluorides and fluorapatites are by-products. Of the fluoride compounds released into the atmosphere, the gaseous forms are most harmful and the particulates the least. Fluorides are more damaging to many species of vegetation than equivalent concentrations of other aerial phytotoxicants.

Analytical methods are available for diagnosis of plants exposed to fluorides. Although these methods are not specific nor do they have the sensitivities to distinguish individual compounds at trace concentrations in the atmosphere, the kinds and relative concentrations of the components can be estimated from theoretical calculations and from emission data from known sources. Pack et al. (1959), Altshuller (1965), Hill (1969), and Weinstein and McCune (1970) discuss methodology of sampling for fluorides.

Symptoms

There is much variation in the production of symptoms by vegetation to fluoride-induced injury. Usually, injury results from a gradual accumulation of fluorides in the plant tissues over a period of time. Therefore, the extent and degree of injury may depend upon species and varietal differences in susceptibility; upon the concentration, form of fluoride, and duration of exposure; and upon the overall modifying aspects of the total environment.

Assessments of the possible effects of fluoride on forest broad-leaved species are meager. Thus, description of symptom manifestations must necessarily be based on the findings of fluoride research on related dicotyledonous species. Ledbetter et al. (1960) have shown that fluorides penetrated through the stomata and accumulated primarily in the tips and margins of the leaf blades. Symptoms from this accumulation of fluorides may either be of the acute form, a high concentration for a short exposure period, or the chronic type, a low-fluoride level for an extended exposure time.

But fluoride dosage, a product of concentration and time, does not always correlate with the intensity of symptom expression in plant injury. Romell (1941) found 22 mg fluoride per mg dry weight in visibly injured leaves, 16 mg in other parts of the same leaves, and 6 mg in healthy foliage of Norway maple. Wander and McBride (1956) were unable to associate the amount of chlorosis on citrus foliage (Citrus species) to fluoride content. Threshow (1969) also could not ascribe any relations between fluoride concentration in the atmosphere and in the leaf nor between the latter and plant injury. Generally, the most sensitive plants are injured after a relatively short exposure, while tolerant ones require a longer treatment at identical fluoride concentrations (Middleton et al. 1965). Thomas (1961) has advanced some suggestions for this range of tolerances of plants to the toxic effects of fluorides.

Weinstein and McCune (1970) have summarized the responses of dicotyledonous plants to fluorides. The initial symptom on broad-leaved species was usually a chlorosis of the leaf tip which later extended downward along the margin and inward toward the midrib. Chorophyll breakdown intensified with additional exposure until the midrib and some veins presented a greenish arborescent design on a mottled background. With more fumigation, the chronic areas exhibited acute injury, became necrotic, dropped out, and this created a notching of the leaves. Longitudinal extension of leaves may be curtailed, and they may present a savoyed or cupped shape.

Treshow (1969) considered characteristic symptoms of fluoride injury on many broad-leaved species to be necrosis predominantly at the leaf tips and margins where toxic compounds mass.

He reported that, on sensitive species of apricot, an opaque, gray-green hydrotic discoloration of marginal tissues was the initial response to fumigation. The water-soaked tissues darkened within 2 or 3 days during hot days. If temperatures remained low and no other stresses were superimposed on fluoride-injured field trees, symptoms could be delayed for several weeks. Under conditions of rapid assimilation of fluorides by the foliage, and without their translocation to the extremities of the leaves, lethal concentrations could build up throughout the blade of the leaf, producing a random pattern of intercostal lesions. The mixture of necrotic and chlorotic markings was characteristic of this kind of fluoride injury.

Apricot leaves exposed to low-fluoride concentrations initially developed semicircular markings 1 to 3 cm in diameter along the leaf margins. A sharply defined narrow band, reddish brown in color, usually separated healthy from diseased tissues. Successive fumigations produced additional necrotic lesions of a wavy, zonate appearance. These necrotic areas can separate and drop off, but rarely do apricot leaves abscise (although DeOng (1946) and Brewer et al. (1960) have seen premature defoliation on some fruit trees).

Threshow and Pack (1970) further claimed that these wavy color bands of necrosis are the most definitive characteristic of fluoride injury on sensitive, broad-leaved plants. But they believed the amount of chlorosis and necrosis can vary among species.

Brewer et al. (1960) observed that older leaves can develop a tolerance and that youngest leaves seem to accumulate fluorides for a time before symptoms occur. On the leaves of citrus, sweet cherry, and poplar, fluorides produced a chlorosis. This mottling extended from the leaf margin inward between the larger veins. With continued exposure, the mottled region turned completely yellow, and the area between chlorotic and green tissue was sharply demarcated. This reaction was common on plants fumigated in the greenhouse. It does not appear too frequently on leaves of field plants, but may be associated in species where necrotic symptoms predominate.

Fumigation with high concentrations of fluorides for short periods may cause acute injury in the form of interveinal and marginal necrosis similar to that produced by sulfur dioxide (Thomas et al. 1952). Haselhoff and Lindau (1903) fumigated oak and chestnut with hydrogen fluoride. On oak leaves, injury was initated at the edge as sharply defined marginal necrotic spots. On chestnut leaves, the discoloration was light brown, and lesions were formed on both sides of the midrib and primary veins.

At times, early symptoms of fluoride injury to walnut were pale green or gray water-soaked lesions surrounded by reddish-brown borders. Later, the injured areas fell out to give an irregular border or shot-hole appearance to the leaves (DeOng 1946).

Other symptoms of fluoride injury may include its effects on tree growth. Brewer et al. (1960) exposed orange trees in a greenhouse to 1 to 5 ppb (parts per billion) hydrogen fluoride for over 2 years. Leaf size, tree height, crown volume, green weight, and linear shoot growth were all reduced. But Adams and Sulzbach (1961) reported that all bean plants fumigated with fluorides for 10 to 20 days had a longer initial growth than control plants. Similar responses are mentioned by Treshow et al. (1967) on Douglas fir where they found that needles exposed to low fluoride concentrations were longer than those on trees grown in the absence of this pollutant. Anderson (1966) showed that field-grown aspen trees exposed to fluorides had their leaf size reduced by 30 percent.

Threshow (1969) believed higher fluoride concentrations to be inhibitory even without the visible symptoms of chlorosis or necrosis. Also, fluorides at sublethal levels have a definite effect on growth. And these responses in growth may be stimulated or checked depending on the fluoride concentrations, the sensitivity of the species, and probably the influence of environmental factors. Therefore, he concluded that fluorides may cause necrosis of the most susceptible plants, growth suppression of less sensitive plants, and growth increases in more tolerant plants, all at the same time of fluoride exposure.

Additional reviews on the absorption, accumulation, and subsequent responses of forest broad-leaved species to fluorides are in the European literature by Garber (1962, 1966), Bossavy (1965), Halbwachs and Kisser (1967), and Dassler (1969).

Mode of Action

Some studies were made on the site of action of absorbed fluorides and their methods of affecting injury in plants. Thomas (1951) presented some of the earlier work and cited inhibition of enzyme systems and cessation of photosynthesis as possible explanations for the mechanism of toxication. Ledbetter et al. (1960) reported respective decreases in the quantity of fluorides in particulate fractions of tomato in the cell wall, chloroplasts, water soluble protein, and mitochondria. Chang and Nickerson (1954) suggested that fluorides disrupted cell wall metabolism through their interference with cell wall polysaccharide synthesis, and also fluorides prevented growth of oat coleoptile sections by inhibition of the enzyme phospho-glucomutose, which is involved with cellulose production (Ordin and Altman 1965).

Susceptibility

The threshold where atmospheric fluoride becomes toxic differs between some hardwood species by orders of magnitude of two or more. Thomas and Hendricks (1956) suggested that for each species there was a concentration below which no curtailment in photosynthesis occurred, but above which chlorophyll production was hindered without any apparent visible injury to leaves. They believed variations in sensitivity could be affected by the interaction between fluorides and specific enzyme

systems, by rate of absorption and translocation of absorbed fluorides to the extremities of the leaf, and by the extent to which inorganic ions in the leaf inactivated fluorides as insoluble substances.

Many researchers have analyzed the atmospheric concentrations of fluorides that can injure tree species in fumigation studies and have classified plants for their susceptibility. These values as specified in the literature are somewhat in doubt because of the undefined experimental procedures, the difficulty in maintaining a constant rate of hydrogen fluoride in chambers, the inability to simulate conditions as exist in field trees, and the lack of knowledge on the nature and effects of the total environment upon the plant species and its injurious pollutant.

Adams et al. (1957) investigated the relationship among exposure periods, foliar burn, and fluorine content of vegetation exposed to hydrogen fluoride. They evolved the term "exposure factor". This was an empirical value defined as the sum of products of daily exposure to hydrogen fluoride and was a measure of the rate of accumulating fluoride by an exposed plant as its threshold of visible injury developed. They cautioned that their greenhouse findings could not be compared to field conditions where susceptibility and environmental factors reacted differently.

Tendron (1964) published a list of trees sensitive to fluorine deriv tives (according to Andre Mazel, France). English walnut, Spanish chestnut (Castanea sativa Mill.), and paulownia (Paulownia species) were categorized as highly sensitive. Intermediate in sensitivity to fluorine compounds were European Hornbeam (Carpinus betulus L.), European ash (Fraximus excelsior L.), English oak, European beech, black poplar (Populus nigra L.), European white birch (Betula pendula Roth), European filbert (Corylus avellana L.), English holly (Illex aquifolium L.), Oriental plane, and black locust. Tolerant species included English elm, black alder (Alnus glutinosa Gaetrn.), American mountain-ash (Sorbus domestica Marsh.), European alder (Sambucus nigra L.), and European linden (Tilia cordata Mill.).

Treshow and Pack (1970) listed a group of common species and their reactions to fluorides based on fumigation studies and personal observations. Their ratings were in general agreement with those published previously by Thomas and Hendricks (1956) and Zimmerman and Hitchcock (1956).

Treshow and Pack (1970) said that their categories of susceptibility were subjective and arbitrary, because species response was dependent on numerous environmental variables. Only boxelder was cited as sensitive to fluorides. Forest broad-leaved species intermediate to fluoride reactions in approximate order of increasing tolerance were English walnut, black walnut, willow, quaking aspen, silver maple, green ash, red mulberry, European linden, and catalpa (Catalpa bignonioides Walt.). Tolerant broad-leaved trees were American linden (Tilia americana L.), American elm, cutleaf birch (B. pendula var. Youngii Rehd.), European mountain-ash, elderberry, plane tree, and Modesto ash (F. velutina Torr.).

Related listings of forest deciduous trees tolerant to fluorinated compounds can also be found in Adams et al. (1957), Bolay and Bovay (1965), and Bossavy (1965) publications.

OZONE

In southern California, Middleton et al. (1950) reported the first incident of photochemical smog injury to vegetation. Richards et al. (1958) elucidated some of the mystery of the smog complex when they identified ozone as the cause of stipple on grape foliage. After this report, Heggestad and Middleton (1959), Ledbetter et al. (1959), Taylor et al. (1960), Daines et al. (1960), Hill et al. (1961), Berry and Ripperton (1963), and Engle et al. (1965) published their findings on the harmful effects of ozone on various plant species.

Ozone is probably involved with more injury to vegetation than any other pollutant in the United States (Hill et al. 1970) and is no longer considered a local problem, but a major threat in all densely populated areas.

Ozone, an active oxidizer, is synthesized photochemically from a cyclic reaction involving nitrogen dioxide and oxygen. Light energy splits a molecule of nitrogen dioxide and releases an atom of oxygen. This atom combines with oxygen to produce ozone, and nitric acid remains. Without any interference, the reaction is reversible, and oxygen and notrogen dioxide are re-formed. The addition of hydrocarbons from automobile exhausts and related pollution sources changes the cyclic reaction and affects an oxidation of the hydrocarbons, which then convert the nitric oxide to nitrogen dioxide, thereby allowing ozone to accumulate.

Sources of injurious ozone are the activities of man, the upper atmosphere, electric storms, and plantings of conifer trees. Photolytic reactions in polluted atmospheres are probably the major sources of ozone. Man's automobiles and industries waft tons of hydrocarbons and oxides of nitrogen into the atmosphere, which are then energized by the rays of sunlight in the presence of oxygen to form ozone.

Along with the combustion of organic fuels as a primary contributor of reactants for phytotoxic oxidants, the outer stratosphere can add large quantities of ozone. Toposperic ozone may be carried to the earth's surfaces by meterological disturbances (Bowen and Regener 1951; Leone et al. 1962), or by jet streams (Wanta et al. 1961). Ozone may also be formed during electric storms and cause injury to susceptible plants.

Another possible source of ozone may be from the action of ultraviolet sunlight on reactive hydrocarbons released by large masses of vegetation. Went (1955) proposed that ozonides and peroxides exist in the natural blue smog over conifer forests. Stephens and Scott (1962) have shown that photochemical reactions between pinene and nitrogen dioxide can produce ozone and peroxyacetyl nitrate. Rasmussen and Went (1965) suggested that light oxidation of these reactive terpenes contributed to air degradation and possible injury to conifer species.

All methods of ozone analysis in the atmosphere are subject to interference both from oxidizing compounds, such as oxides of nitrogen, and from reducing compounds of sulfur dioxide. Automatic analyzers were developed for computing total oxidant concentrations based on either colorimetric or coulometric measurements of liberated iodine, but contamination of oxidant pollutants curtail the reliability of these instruments.

Currently, an absolute measurement of ambient ozone is not feasible, because of the reactions of other oxidants upon potassium iodide. Therefore, all readings are referred to as indices of oxidants, and types of analysis and instrumentation should be mentioned in reporting levels of ambient ozone. Further references on standards for sampling and analysis of total oxidants can be obtained in the publications by Altshuller (1965) and Pack and Adams (1966).

Symptoms

Many factors affect the responses of forest broad-leaved trees to ozone. Variability in response can occur between hybrid clones of the same parentage or on leaves of the same branch, probably because of the physiological disposition and genetical susceptibility of the host as influenced by its total environment.

The visible symptoms of ozone injury to plants were classified by Hill et al. (1970) into four general types as pigmented lesions, surface bleaching, bifacial necrosis, and chlorosis. Usually one type of injury occurred, but two or more types of markings may be seen on a single leaf or on different plants within the same area. On deciduous trees, leaves displayed little injury initially, but became chlorotic a few days after ozone exposure, followed by a premature loss of some of the mottled foliage.

Pigmented and bleached markings were the most common type of injury observed on the upper surfaces of the foliage of hardwood trees. Individual lesions normally appeared as small, discrete spots of pigmented, chlorotic, or necrotic tissues when viewed with a hand lens. Bleached lesions can range from a few to an infinite number of multicolored spots. When the lesions were numerous, an overall bronzed, silvery, or purple cast was imparted to the upper leaf surface. Some leaves displayed only a slight loss in green color; but bifacial necrosis on the foliage of other species, such as aspen, was commonly the result of ozone fumigation.

Hibben (1969) exposed sugar maple trees to ozone under laboratory and field conditions. Ozone injury to field trees was obvious within 12 hours after treatment when single and multiple groups of parenchyma cells displayed a hydrotic, yellow-green, or brown condition. Two days after fumigation, minute flecks were visible in the vein islets. These markings were not sunken and were marginless, as often seen with leaf spots from foliar fungi. They were randomly distributed in the interveinal tissues within 1 to 2 mm of the primary and secondary veins,

but sometimes a reticulate pattern of flecks was concentrated along the veins. Both single flecks and multiple patterns of injury occurred on a single leaf or on leaves of the sample branch. Vascular elements around the vein islets often were discolored and the underside of a fleck appeared water soaked.

Higher concentrations of ozone produced a bifacial necrosis that injured the interveinal tissues. Some leaves were flecked over most of the blade, but interveinal browning occurred at the base. Both flecking and necrosis were visible on the same limb. Leaf drop only occurred on those branches most severely injured. Under controlled conditions in the laboratory, similar responses were encountered on exposed sugar maple trees, except that the flecks were usually grayish white or tan instead of a reddish brown as on field trees. Twig dieback never developed in any ozone fumigations.

Wood and Davis (1969) conducted similar ozone fumigation studies on 16 deciduous tree species. On these hardwoods, the most common symptoms were stipple, chlorosis, tissue collapse, and necrosis. Necrotic stippling of the upper surface of the leaf was the most common symptom. Stipples were minute in size and varied from light tan to dark brown to purple brown in color. Aside from the ubiquitous occurrence of stippling on hardwood species, they found that the development of other symptoms depended on the concentration of ozone, temperature, and relative humidity during fumigation, and the species being ozonated.

Wood et al. (1969) observed the following symptoms on the various 2- to 5-year-old hardwood seedlings fumigated with ozone. On green ash, 24 hours after treatment, a dark-brown, interveinal stipple of the upper leaf surface was common. At times, the discolored areas coalesced to produce a smoky appearance of the leaves. Tissue collapse and necrotic spots were associated with stippling.

Similar stippling occurred on white ash. At first, stipples were hydrotic and dark, but later they became white to light brown to purplish brown. Size and distribution of the stipples varied on individual leaflets. Bifacial necrotic spots were also present and at times were surrounded by a purple halo. On sensitive trees exposed to high concentrations of ozone, tissue collapse of the marginal and tip areas appeared about a day after fumigation. The color became a light brown and the injured parts appeared transparent.

Sweetgum showed a purplish-brown stippling of the entire upper surface of the leaf. These stipples were larger than those on the ash species, but fewer were present per unit area of leaf. All leaves of a seedling were similarly affected.

Dark-brown stipples were evident on the upper surface of leaves within a few hours after fumigation of pin oak plants. Subsequently, these stipples and associated light- to dark-brown colored spots coalesced to form necrotic areas.

Light-brown to black stipples of the upper portion of the leaf were observed on scarlet oak seedlings. Interveinal chlorosis and necrosis were present too. Scarlet oak was one of a few species that indicated an increase in symptoms as its sensitivity intensified with the maturity of the foliage.

On white oak plants, dark-brown to black interveinal stipple was generally visible on the upper surface of leaves. As the trees aged, similar symptoms developed, but they were more apparent on the lower half on the plants and on the lower leaf surface. Often, within 2 weeks after fumigation, heavily stippled areas became necrotic.

On yellow-poplar seedlings, a dark-brown to black stipple of the upper leaf surface was most common. But, unlike the other hardwoods, symptoms also developed on the lower surfaces. Within a week to 10 days after the various fumigations, leaves with the most severe injury developed an overall chlorosis of tissues followed by necrosis and finally by a partial defoliation of the tree. Yellow-poplar, 14 weeks after bud break, produced prominent stipples at the bases of the petioles on foliage from the lower half of the trees.

Physiological effects on growth and yield reductions because of ozone fumigation are reported by Taylor (1968), Thompson and Taylor (1969), and Feder and Campbell (1968). More research and additional assessment of these findings must be accomplished before an evaluation can be made of the biochemical and physiological responses of field plants to ozone.

Mode of Action

Several theories are proposed for the injury of plants by ozone. Freebairn (1959) observed ozone to inhibit the oxygen uptake associated with the mitochondrial citric acid activity. He believed that ozone acted as a highly specialized inhibitor and probably did not act by damaging the cellular membrane. Giese and Christensen (1954) suggested that ozone acted by destroying cellular membrances. Siegel and Porto (1961) considered ozone injury as a kind of oxygen stress mechanism, and they enlarged on a theory relating growth to an internal balance between oxidants and antioxidants. Taylor et al. (1961) proposed that the growth suppression in citrus trees was due to the inhibition of photosynthesis.

Susceptibility

Little is known on the susceptibility of forest broad-leaved trees to ozone. Considerable variation in varietal and species responses to this oxidant gas has been observed. Genetic susceptibility and environmental influences should be determined for each interaction of host and pollutant.

Wood and Davis (1969) found that many hardwood species are sensitive to 25 pphm (parts per hundred million) of ozone for 4 hours. Species sensitive to an ozone exposure of 25 pphm for 8 hours were green ash, white ash, sweetgum, pin oak, scarlet oak, white oak, and tulip-poplar. Hardwood trees responding to fumigations of 25 pphm in ozone for 4 hours included green ash, white ash, white oak, and tulip-poplar. White ash was the only species injured by ozone for 10 pphm for 8 hours. Forest broad-leaved species tolerant to 25 pphm of ozone for 8 hours were European white birch, little leaf linden, Norway maple, sugar maple, English oak, red oak, and shingle oak.

Hardwood species injured by field or laboratory fumigation with ozone are listed by Hill et al. (1970) to be alder, quaking aspen, boxelder, catalpa, honey locust, silver maple, Gambel oak, sycamore, and weeping willow.

CONCLUSIONS

Community air pollution is a daily problem. The foul breath of dirty air as a regional or perhaps as a global threat has also had serious repercussions on vegetation and human welfare. These problems may occur from unfavorable meteorlogical conditions and when air resources are utilized faster than the natural processes are able to replenish them. And as society continues to expand and release new forms of waste materials into our contaminated aerial environment, we must expect a confrontation with more kinds and numbers of air pollution episodes in the immediate future.

In the United States, long-range forecasts by Heller and Walters (1965) indicate an expectancy for a doubling in population and a threefold increase in needs for energy use. Ehrlich (1970) said that in the next 15 years, U. S. power companies will construct as many coal- and oil-fired power generating plants as exist now. Little (1970) assessed the air pollution potential from waste incinerators and projected a 400-percent increase in the plastics component of waste could multiply hydrogen chloride effluents from halogenated polymers by 5-1/2 times within the next 30 years. Similar raises could be anticipated in the release of sulfur dioxide, oxides of nitrogen, and particulate matter.

Future trends indicate that the injurious effects of air pollutants to forest plantings will continue and intensify in future decades. Also, new physical and chemical forms of pollution may arise from expanding technology. And some of the currently recognized minor pollutants may become more destructive as their sources are utilized and as they become more prevalent. There is a need now to anticipate and plan a program of research on the nature of air pollution symptomatology to forest broad-leaved trees.

More emphasis and consideration is necessary for the diagnosis of air pollution injury to trees. Interpreting the cause of a disease and the contributing environmental effects of this relationship can be most difficult in any evaluation of a pathogenic or physiogenic disturbance. Initial observations can be effectively accomplished in controlled experiments; but because these findings must relate to field problems, the final estimates must be made under field conditions. Nevertheless, accurate atmospheric analyses that characterize specific pollutants under controlled environmental conditions may eventually permit more understanding of the complexity of the mixture of pollutants in the atmosphere and their effects on vegetation.

More effort is needed for the development and improvement in methodology of plant and atmospheric analyses. Deficiencies exist in analytic methods and in instrumentation for accurate determination of the responsible toxicants. Until these difficulties are resolved, the best method of analysis and the best equipment should be used, and their faults should be evaluated, and the experimental findings should be discussed in relation to the limitations and interferences involved in the analysis of injurious substances.

The previous suggestions of research direction in further elucidating symptomatology of pollution-affected forest trees are generally recognized and of immediate concern to most investigators. But in addition to these obvious recommendations that must be resolved, specific studies, both of an applied and basic nature, can be considered in delineating symptoms of harmful effects of air pollutants to forest broad-leaved species. Studies may consist of work on (1) synergistic effects after long-term, low-level exposure; (2) patterns of abnormal growth from ambient levels of fumigation; (3) acute and chronic symptoms associated with high, low, and intermittent treatment; (4) comparison of symptoms from controlled fumigation with specific air pollution injury observed in areas of high industrial fume emissions; (5) interaction of environmental conditions and host responses to pollutants; (6) biochemical action of pollutants on nature of host resistance; and (7) description of pollutant dosage in relation to symptom expression.

Considerable research effort will be required to supplement the information already realized about the responses of forest broad-leaved trees to gaseous and particulate pollutants. When the complex, interrelated tree-environmental factors are more completely understood, accepted standards of methodology will be established. These accepted standards for atmospheric analyses will permit a more realistic appraisal of the magnitude of air pollution episodes and thus serve as a means for estimating economic losses, determining impact on forest resources, and recommending control criterion for air quality.

LITERATURE CITED

ACATAY, A. 1968. Smoke damage from the copper-smelting works in Murgul. Istanbul Univ. Orm. Fak. Derg. 18A: 1-17.

ADAMS, D. F., J. W. HENDRIX, AND H. G. APPLEGATE. 1957. Relationship among exposure periods, foliar burn, and fluorine content of plants exposed to hydrogen fluoride. Agr. and Food Chem. 5: 108-116.

_____, AND C. W. SULZBACK, 1961. Nitrogen deficiency and fluoride susceptibility of bean seedlings. Science 133: 1425-1426.

ALTSHULLER, A. P. 1965. Air pollution. Anal. Chem. 37: 11-20.

ANDERSON, F. K. 1966. Air pollution damage to vegetation in Georgetown Canyon, Idaho. M.S. Thesis., Univ. of Utah. 102 p.

ANONYMOUS. 1968. Plants absorb sulfur dioxide release hydrogen sulfide. Sulfur Inst. J. 4: 17.

ANTIPOV, V. G. 1957. The influence of fumes from industrial works on the seasonal development of trees and shrubs. Bot. Z. 42: 92-95.

BARRETT, T. W., AND H. M. BENEDICT. 1970. Sulfur dioxide. In J. J. Jacobson and A. C. Hill [ed.]. Recognition of air pollution injury to vegetation: a pictorial atlas. Inf. Rep. No. 1, Sect. D, 1-17. Herbick and Held Printing Co., Pittsburgh.

BEREND, I., AND E. CSUTI. 1959. Smoke and dust damage in agriculture. Novenyved Idosz. Kerd. 3: 47-53.

BERRY, C. R., AND L. A. RIPPERTON. 1963. Ozone, a possible cause of white pine emergence tipburn. Phytopathology 53: 552-557.

BOLAY, A., AND E. BOVAY. 1965. The sensitivity to fluorinated gases of some plant species of the Valais. Phytopathology Z. 53: 289-298.

BOSSAVY, J. 1965. Scale of sensitivity to fluorine. Rev. Franc. 17: 205-211.

_____. 1965a. Leaf necrosis caused by fluorine. Rev. Franc. 17: 801-811.

BOWEN, G. J., AND V. REGENER. 1951. On the automatic chemical determination of atmospheric ozone. J. Geophys. Res. 56: 307-324.

BRANDT, C. S., AND W. W. HECK. 1968. Effects on air pollutants on plants. In A. C. Stern [ed.]. Air pollution, 1: 401-443. Acad. Press, New York.

BREWER, R. F., F. H. SUTHERLAND, F. B. GUILLEMET, AND R. K. CREVELING. 1960. Some effects of hydrogen fluoride gas on bearing navel orange trees. Proc. Amer. Soc. Hort. Sci. 76: 208-214.

CHANG, C. W., AND W. J. NICKERSON. 1954. Polysaccharide synthesis in growing yeasts. J. Biol. Chem. 208: 395-407.

DAINES, R. H. 1968. Sulfur dioxide and plant response. J. Occup. Med. 10: 84-92.

_____, I. A. LEONE, AND E. BRENNAN. 1952. The effect of fluoride on plants as determined by soil nutrition and fumigation studies. In L. C. McCabe [ed.]. Air pollution. Proc. U. S. Tech. Conf. Air Pollut. 97-105. McGraw-Hill, N. Y.

_____, I. A. LEONE, AND E. G. BRENNAN. 1960. Air pollution and its effects on agriculture in New Jersey. New Jersey Agr. Exp. Sta. Bull. 794: 1-14.

DASSLER, H. G. 1963. Problems of research on fume damage to forests. Biol. Zbl.82: 217-228.

_____. 1969. The fluorine content of plants in air-polluted and fume-free areas. Flora Jena. 159A: 471-476.

DEONG, E. R. 1946. Injury to apricot by fluorine deposit. Phytopathology 36: 469-471.

DIMITROVSKY, K. 1965. The effect of industrial fumes on forestry in lignite-mining areas. Lesn. Prace 44: 314-316.

DOCHINGER, L. S. 1968. The impact of air pollution on eastern white pine: the chlorotic dwarf disease. J. Air Pollut. Contr. Assoc. 18: 814-816.

_____, AND C. E. SELISKAR. 1970. Air pollution and the chlorotic dwarf disease of eastern white pine. Forest Sci.16: 46-55.

DORRIES, W. 1932. Uber die Brauchbarkeit der spectroscopischen Phaophytinprobe in der Rauchschaden-Diagnostik. Z. Pflanzenkrankh. u. Gallenkunde 42: 257-273.

DREISINGER, B. R., AND P. C. MCGOVERN. 1970. Monitoring atmospheric sulfur dioxide and correlating its effects on crops and forests in the Sudbury area. In Impact of air pollution on vegetation conference. Toronto, Ontario Sect. and TR-7 Agr. Comm. APCA Sess. 1: 1-23.

EHRLICH, S. 1970. Air pollution control through new combustion processes. Environ. Sci. Technol. 4: 396-400.

ENGLE, R. L., W. H. GABLEMAN, AND R. R. ROMANOWSKI. 1965. Tip burn, an ozone incited response in onion, Allium cepa L. Proc. Amer. Soc. Hort. Sci. 86: 468-474.

FEDER, W. A., AND F. J. CAMPBELL. 1968. Influence of low level of ozone on flowering of carnations. Phytopathology 58: 1038-1039.

FREEBAIRN, H. T. 1959. The toxicity of ozone, a constituent of smog. J. Appl. Nutr. 12: 1-13.

GARBER, K. 1962. Absorption of toxic substances through the bark of trees. Wiss. Z. Tech. Univ. of Dresden 11: 549-552.

_____. 1963. The effect on plants of fluorine-containing waste gases. Landev. Forsch. Sonderh. 17: 20-25.

_____. 1966. The influence on vegetation of an F-polluted atmosphere. Angew. Bot. 40: 12-21.

GIESE, A. C., AND E. CHRISTENSEN. 1954. Effects of ozone on organisms. Physiol. Zool. 27: 101-115.

GISIGER, L. 1955. Fluorine injury in the Rhunfelden and Mohlin districts. Mitt. Schweiz. Land wirtsch 3: 81-91.

GORDON, A. G., AND E. GORHAM. 1963. Ecological aspects of air pollution from an iron-sintering plant at Wawa, Ontario. Can. J. Bot. 41: 1063-1078.

HALBWACHS, G., AND J. KISSER. 1967. Dwarfed growth in Norway spruce and birch caused by smoke pollution. Clb. ges. Forstw. 84: 156-173.

HASELHOFF, E., G. BREDEMANN, AND W. HASELOFF. 1932. Entstehung und Beurteilung von Rauchschaden. Verlagsbuchhandlung Gebulder Berntreger. Berlin.

_____, AND G. LINDAU. 1903. Die beschadigung der vegetation durch rauch. Gebruder Borntraeger. Leipzig. 412 p.

HAYWOOD, J. K. 1905. Injury to vegetation by smelter fumes. U. S. Dep. Agr. Bur. of Chem. Bull. 89: 1-23.

_____. 1910. Injury to vegetation and animal life by smelter wastes. U. S. Dep. Agr. Bur. of Chem. Bull. 113: 1-63.

HECK, W. W. 1968. Factors influencing expression of oxidant damage to plants. Ann. Rev. Phytopathol. 6: 165-188.

HEDGCOCK, G. G. 1912. Winter killing and smelter injury in the forests of Montana. Torreya 12: 25-30.

_____. 1914. Injuries by smelter smoke in southeastern Tennessee. Wash. Acad. Sci. J. 4: 70-71.

HEGGESTAD, H. E., AND J. T. MIDDLETON. 1959. Ozone in high concentrations as a cause of tobacco leaf injury. Science 129: 208-210.

HIBBEN, C. R. 1969. Ozone toxicity to sugar maple. Phytopathology 59: 1423-1428.

HILL, A. C. 1969. Air quality standards for fluoride vegetation effects. J. Air Pollut. Contr. Assoc. 19: 331-336.

_____, H. E., HEGGESTAD, AND S. N. LINZON. 1970. Ozone. In J. J. Jacobson and A. C. Hill [ed.]. Recognition of air pollution injury to vegetation: a pictorial atlas. Inform. Rep. No. 1, Sect. B, 1-22. Herbick and Held Printing Co., Pittsburgh.

_____, M. R. PACK, M. TRESHOW, R. J. DOWNS, AND L. G. TRANSTRUM. 1961. Plant injury induced by ozone. Phytopathology 51: 356-363.

HOCHHEISER, S. 1964. Methods of measuring and monitoring atmospheric sulfur dioxide. U. S. Public Health Serv. Pub. 999-AP6: 1-47.

HOLTE, W. 1961. Fluorine damage to agricultural and garden plants from fertilizer factories. Ber. Landesants. Bodenmutzungschutz Nordrhein- Westfalen. p. 46-62.

KATZ, M. 1949. Sulfur dioxide in the atmosphere and its relation to plant life. Ind. Eng. Chem. 41: 2450-2465.

_____, G. A. LEDINGHAM, AND A. W. MCCALLUM. 1939. Symptoms of injury on forest and crop plants. In Effects of sulfur dioxide on vegetation. Nat. Res. Counc. Can. (Chapt.) 3: 51-103.

_____, AND A. W. MCCALLUM. 1952. Effects of sulfur dioxide on conifers. In L. C. McCabe [ed.]. Air pollution. Proc. U. S. Tech. Conf. Air Pollut. p. 84-96. McGraw-Hill, N. Y.

KNABE, W. 1966. Rauchschadenforschung in Nordameuka. Forstarchiv 37: 109-119.

LEDBETTER, M. C., R. MAVRODINEANU, AND A. J. WEISS. 1960. Distribution studies of radioactive fluorine-18 and stable fluorine-19 in tomato plants. Contrib. Boyce Thompson Inst. 20: 331-348.

_____, P. W. ZIMMERMAN, AND A. E. HITCHCOCK. 1959. The histopathological effects of ozone on plant foliage. Contrib. Boyce Thompson Inst. 20: 275-282.

LEONE, I. A., E. BRENNAN, AND R. H. DAINES. 1962. Daily fluctuations of phytotoxic air pollutants in three New Jersey communities as influenced by certain meteorlogical parameters. Plant Disease Rep. 46: 140-144.

LINZON, S. 1965. Sulphur dioxide injury to trees in the vicinity of petroleum refineries. Forest. Chron. 41: 245-250.

_____. 1969. Symptomatology of sulphur-dioxide injury on vegetation. In N. L. Lacasse and W. J. Moroy [ed.]. Handbook of effects assessment vegetation damage. Penn. State Univ. Sect. 8, 1-13. Univ. Park, Penn.

LITTLE, A. D. 1970. Air pollution from incinerators will be continuing problem. Environ. Sci. Tech. 4: 718.

MACINTIRE, W. H. 1952. Air versus soils as channels for fluorine contamination of vegetation of two Tennessee locales. In L. C. McCabe [ed.]. Air pollution. Proc. U. S. Tech. Conf. Air Pollut. p. 53-58. McGraw-Hill, N. Y.

MAHYRHOFER, J. 1893. Uber pflanzenbeschadigung, veranlasst durch den betreib einen superphosphatfabrik. Z. Pflanzenbrankh. 3: 50-51.

MIDDLETON, J.T., L.O. EMIK, AND O.C. TAYLOR. 1965. Air quality criteria and standards for agriculture. J. Air Pollut. Contr. Assoc.15: 476-480.

_____, J. B. KENDRICK, AND H. W. SCHWALM. 1950. Injury to herbaceous plants by smog or air pollution. Plant Disease Rep.34: 245-252.

NIKOLAEVSKĬĬ, V. S. 1968. Activity of certain enzymes and the gas resistance of woody plants. Trud. Inst. Ekol. Rast. Zivot. Ural skŭĭ Fil. ANSSSR 62: 208-211.

NOVAK, K. 1929. Damage to vegetation from gases in smoke. Z. Angew. Chem. 42: 123-126.

NOZICKA, J. 1963. Damage caused by smoke in Czechoslovakian forests and its control up to 1918. Prace Vyzkum. Ust. Lesn. CSSR 26: 237-258.

OKANOUE, M. 1958. On an injury to Akamatsu Forest in the vicinity of the smelting works at Hitachi Ringyo Shikensho Kenkyu Hokoku 105: 141-147.

ORDIN, L., AND A. ALTMAN. 1965. Inhibition of phosphoglucomutase in oak coleoptiles by air pollutants. Physiol. Plant. 18: 790-797.

OST, H. 1907. Der kamp gegen schadliche industriegase. Z. Angew. Chem. 20: 1689-1693.

PACK, M. R., AND D. F. ADAMS. 1966. Problems of relating atmospheric analyses to effects of air pollution on agriculture. J. Air Pollut. Contr. Assoc. 16: 219-224.

_____, A. C. HILL, M. D. THOMAS, AND L. G. TRANSTRUM. 1959. Determination of gaseous and particulate inorganic fluorides in the atmosphere. ASTM Spec. Tech. Pub. 281: 27-44.

PELZ, E. 1956. Gaseous air pollution and choice of species in districts with industrial smoke hazards. Forst u Jagd. 6: 347-349.

_____, H. BEYER, AND G. BLEYER. 1963. A study of diagnostic methods and effects of smoke damage in the neighborhood of a lead works. Wiss. Z. Tech. Univ. Dresden 12: 209-216.

PROKOPIEV, E. 1965. Damage by sulphur gases in forest plantations near the G. Damjanov' copper combine. Gorskostop. 2: 3-13.

RASMUSSEN, R. A., AND F. W. WENT. 1965. Volatile organic material of plant origin in the atmosphere. Proc. Nat. Acad. Sci. 53: 215-224.

RHODE, A. 1895. Schadigung von roggenfeldern, durch die einer superphosphat-fabrik enstromenden gase. Z. Pflanzenkrankh. 5: 135-136.

RICHARDS, B. L., J. T. MIDDLETON, AND W. B. HEWITT. 1958. Air pollution with relation to agronomic crops: V. oxidant stipple of grape. Agron. J. 50: 559-561.

ROMELL, L. G. 1941. Localized injury to plant organs from hydrogen fluoride and other acid gases. Svensk. Bot. Tidskr. 35: 271-286.

SCARINGELLI, F. P., B. E. SALTZMAN, AND S. A. FREY. 1967. Spectrophotometric determination of atmospheric sulfur dioxide. Anal. Chem. 39: 1709-1719.

SCHEFFER, T. C., AND G. C. HEDGCOCK. 1955. Injury to northeastern forest trees by sulfur dioxide from smelters. U. S. Dep. Agr. Forest Serv. Tech. Bull. 1117: 1-49.

SCHROEDER, J., AND C. REUSS. 1883. Die beschadigung der vegetation durch rauch und die oberharzer huttenrauchschaden. Berlin. 333 p.

SCURFIELD, G. 1960. Air pollution and tree growth. Forest. Abstr. 21: 339-347, 517-528.

SETTERSTROM, C., AND P. W. ZIMMERMAN. 1939. Factors influencing susceptibility of plants to sulphur dioxide injury. Contrib. Boyce Thompson Inst. 10: 155-181.

SIEGEL, S. M., AND F. PORTO. 1961. Oxidants, antioxidants, and growth regulation. In R. M. Klein [ed]. Plant growth regulations. p. 341-353. Iowa State Univ. Press, Ames.

SPIERINGS, F. 1967. Method for determining the susceptibility of trees to air pollution by artificial fumigation. Atmos. Environ. 1: 205-210.

STEPHENS, E. R., AND W. E. SCOTT. 1962. Relative reactivity of various hydrocarbons in polluted atmospheres. Proc. Amer. Petrol. Inst. 42: 665-670.

TAYLOR, O. C. 1968. Effects of oxidant air pollutants. J. Occup. Med. 10: 485-492.

_____, W. M. DUGGER, E. A. CARDIFF, AND E. F. DARLEY. 1961. Interaction of light and atmospheric photochemical products ('smog') within plants. Nature 192: 814-816.

_____, E. R. STEPHENS, E. F. DARLEY, AND E. A. CARDIFF. 1960. Effects of airborne oxidants on leaves of pinto bean and petunia. Proc. Amer. Soc. Hort. Sci. 75: 435-444.

TENDRON, M. 1964. Effects of air pollution on animals and plants. In European Conference on Air Pollution. Counc. of Europe. Strasbourg. p. 27-69.

THOMAS, M. D. 1951. Gas damage to plants. Ann. Rev. Plant Physiol. 2: 293-322.

_____. 1956. The invisible injury theory of plant damage. J. Air Pollut. Contr. Assoc. 5: 205-208.

_____. 1961. Effects of air pollution on plants. In Air pollution. World Health Organ. Monogr. Ser. 46: 233-278. Columbia Univ. Press, N. Y.

_____, AND R. H. HENDRICKS. 1956. Effect of air pollutants on plants. In P. L. Magil [ed.]. Air pollution handbook. p. 1-44. McGraw-Hill, N. Y.

_____, R. H. HENDRICKS, AND G. R. HILL. 1950. Sulfur metabolism of plants: effect of sulfur dioxide on vegetation. Ind. Eng. Chem. 42: 2231-2235.

_____, R. H. HENDRICKS, AND G. R. HILL. 1952. Some impurities in the air and their effects on plants. In L. C. McCabe [ed.]. Air pollution. p. 41-47. McGraw-Hill, N. Y.

THOMPSON, C. R., AND O. C. TAYLOR. 1969. Effects of air pollutants on growth, leaf drop, fruit drop, and yield of citrus trees. Environ. Sci. Technol. 3: 934-940.

TRESHOW, M. 1969. Symptomatology of fluoride injury on vegetation. In N. L. Lacasse and W. J. Moroz [ed.]. Handbook of effects assessment vegetation damage. Penn. State Univ. Sect. 7, 1-41. Univ. Park, Penn.

_____, F. K. ANDERSON, AND F. M. HARNER. 1967. Responses of Douglas fir to elevated atmospheric fluoride. Forest Sci. 13: 114-120.

_____, AND M. R. PACK. 1970. Fluoride. In J. J. Jacobson and A. C. Hill [ed.]. Recognition of air pollution injury to vegetation. A pictorial atlas. Inform. Rep. No. 1, Sect. D, 1-17. Herbick and Held Printing Co., Pittsburgh.

WANDER, F. W., AND J. J. MCBRIDE. 1956. A chlorosis produced by fluorine on citrus in Florida. Sci. 123: 933-934.

WANTA, R. C., W. B. MORELAND, AND H. E. HEGGESTAD. 1961. Tropospheric ozone: an air pollution problem arising in the Washington, D. C. metropolitan area. Mon. Weather Rev. 89: 289-296.

WEINSTEIN, L. H., AND D. C. MCCUNE. 1970. Effects of fluorides on vegetation. In Impact of air pollution on vegetation conference. Toronto, Ontario Sect. and TR-7 Agr. Comm. APCA Sess. 2, 1-31.

WENT, F. W. 1955. Air pollution. Sci. Amer. 192: 63-70

WENTZEL, K. F. 1956. The part played by industrial and domestic smoke in damage to forest by fumes and in atmospheric pollution. Forstarchiv 27: 84-89.

_____. 1961. Model investigation of local smoke damage to forests. Forstiv. Landesaussch Landw. Forsch. Landes Nordrhein-Westfalen 4: 163-170.

_____. 1968. Susceptibility of plants to air pollution, and differences in resistance. Forstarchiv 39: 189-194.

WHITBY, G. S. 1939. The effects of sulfur dioxide on vegetation. Chem. Ind. 58: 99.

WISLICENUS, H. 1898. Resistenz der fichte gegen saure rauchgase bei ruhender und bei tatiger assimilation. Tharandt. Forstl. Jahrb. 48: 152-172.

WOOD, F. A. 1967. Air pollution and shade trees. Int. Shade Tree conf. 43: 66-82.

_____, J. B. COPPOLINO, D. DAVIS, D. DRUMMOND, AND R. WILHOUR. 1969. Air pollution effects. NAPCA Interim Progr. Rep. AP 00436. p. 1-81.

_____, AND D. DAVIS. 1969. Sensitivity to ozone determined for trees. Sci. Agr. 17: 4-5.

ZAHN, R. 1963. The influence of various environmental factors on plant sensitivity towards sulphur dioxide. Z. Pflanzenkrankh. 70: 81-95.

ZIMMERMAN, P. W., AND A. E. HITCHCOCK. 1956. Susceptibility of plants to hydrofluoric acid and sulfur dioxide gases. Contrib. Boyce Thompson Inst. 18: 263-279.